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# Environmental Risk Factors of Childhood Asthma in Urban Centers

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Asthma morbidity and mortality are disproportionately high in urban centers, and minority children are especially vulnerable. Factors that contribute to this dilemma include inadequate preventive medical care for asthma management, inadequate asthma knowledge and management skills among children and their families, psychosocial factors, and environmental exposure to allergens or irritants. Living in substandard housing often constitutes excess exposure to indoor allergens and pollutants. Allergens associated with dust mites (DM) and cockroaches (CR) are probably important in both onset and worsening of asthma symptoms for children who are chronically exposed to these agents. Young children spend a great deal of time on or near the floor where these allergens are concentrated in dust. Of children (2 to 10 years of age) living in metropolitan Washington, DC, 60% were found to be sensitive to CR and 72% were allergic to DM. Exposure to tobacco smoke contributes to onset of asthma earlier in life and is a risk factor for asthma morbidity. Since disparity of asthma mortality and morbidity among minority children in urban centers is closely linked to socioeconomic status and poverty, measures to reduce exposure to environmental allergens and irritants and to eliminate barriers to access to health care are likely to have a major positive impact. Interventions for children in urban centers must focus on prevention of asthma symptoms and promotion of wellness. — *Environ Health Perspect* 103(Suppl 6):59–62 (1995)

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## Introduction

Asthma morbidity and mortality are disproportionately high among African American (AA) children who live in urban areas, especially those of relatively low socioeconomic status (SES). Inadequate preventive medical care for asthma management and lack of asthma knowledge and management skills among children and their families are significant contributing factors to this problem. Asthma mortality is 5 to 7 times higher for AA children than their white counterparts (1) and they are twice as likely to be hospitalized for asthma (2). Recent studies suggest that the disparity of mortality and morbidity are closely associated with SES, and that poverty rather than race or ethnicity is a more significant risk factor (2,3). AA children have a 20% higher prevalence of asthma than Caucasian children.

There are a number of studies that clearly demonstrate that asthma mortality and morbidity are worse in urban centers

than elsewhere. For example, in Cook County, Illinois, a disproportionately high percent of asthma deaths occurred in the inner city (4) and in East Harlem the rate was 10 times that of the average rate in the United States (5). Hospitalization rates for asthma show a similar pattern. Between 1982 and 1986 hospitalizations for asthma in different neighborhoods of New York City showed the highest rate (115/10,000 population) to be in East Harlem and was 16 times higher than that of the lowest rate (7.2/10,000), which occurred in Greenwich Village–SoHo in Manhattan (5). In Maryland, excess hospitalization rates were noted in children living in the inner city (2).

Disparity of asthma morbidity that exists in urban centers reflects to a great degree the gap that exists in SES and accessibility of health care (especially preventive care) in patients of relatively low SES. For example, 50% of African-American and 42% of Hispanic children are poor (6), and regular health care (when present) for the poor is more likely to be in an emergency room (ER) (7). Insurance status is often a strong predictor of access to care in low income populations (8). Case-controlled studies have identified some risk factors that are associated with increased asthma mortality and morbidity: hospitalization(s) in the recent past; frequent ER visits; previous life-threatening episodes of asthma; inadequate or delayed treatment of acute attacks; labile or deteriorating lung function; cultural or economic barriers to

health care (9). Other risk factors are associated with problems that are characteristic of poverty and the urban center, and day to day life stresses pose barriers to seeking regular and preventive care.

In some cases asthma of children living in urban centers is very difficult to manage. This may due to intrinsic or extrinsic factors. An example of intrinsic factors may be abnormal lung development and function (e.g., premature birth). Extrinsic factors may include the result of chronic exposure to allergens or irritants or psychosocial problems. Many children in urban centers live in substandard housing where exposure to indoor allergens and pollutants (especially tobacco smoke) constitutes a risk factor that can be controlled to a limited degree. In urban dwellings, allergens associated with dust mites (DM) and cockroaches (CR) are believed to be important in both onset and worsening of asthma symptoms. For example, it has been shown that exposure to high levels of mite allergen (in dust) during infancy or in the first year of life results in the increased likelihood of sensitization and positive skin tests to these allergens and the development of asthma (below). Conversely, when mite allergen levels are significantly reduced in the home, the frequency of asthma symptoms is reduced.

One of the earliest reports suggesting a role for CR in asthma was that of a study 30 years ago by Bernton and Brown (10). In a recent survey in our clinics at Howard

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University Hospital in Washington, DC 60% of children (2–10 years of age) were positive to CR (11). This rate of sensitization to CR was comparable to that (58%) found in an atopic population in Chicago where bronchial challenges were also done (12). Reductions in pulmonary function following challenge with CR were noted and strongly suggested a causative role of CR in asthma symptoms in this urban population.

Of urban subjects with asthma and positive skin reactivity to CR antigen, 91% had immediate positive bronchial reactivity, and 48% had late-phase reactions (13). The prevalence of CR allergy among asthma patients is directly related to socioeconomic status and CR allergy in asthmatics of low SES is common, ranging from 52 to 78% (11). The potential importance of CR asthma is underscored by the postulation that recurrent exposure to allergens may be responsible for the airway inflammation in asthma (14).

In general, children with asthma who are exposed to passive tobacco smoke (TS) have been shown to have increased emergency room visits, impaired lung function, and a higher requirement for medications. There is evidence that there is a dose response between the number of cigarettes smoked and the length of time of smoking with the severity of asthma symptoms in children. Exposure to TS contributes to onset of asthma earlier in life and is a risk factor for frequent ER visits.

The health effects of environmental tobacco smoke are being increasingly identified and the effect of environmental TS on respiratory function and lung disease in children has been a particularly intense area of investigation. Tobacco smoke is ubiquitous in the workplace, public and private establishments, and in the home, making exposure to environmental TS virtually unavoidable. In a study of nonsmokers and former smokers, 63.3% of nonsmokers reported some daily exposure, 34.5% reported exposure of at least 10 hours per week and 15.9% reported at least 40 hours of exposure per week (15). About 70% of children in the United States live in homes where there is at least one adult smoker (16).

There is a consistently observed effect of environmental TS from parental smoking on a number of acute childhood respiratory illnesses. Infants of smoking mothers have a greater number of hospital admissions for pneumonia and bronchitis than children of nonsmoking parents (17), and

are more likely to have pneumonia and bronchitis during the first year of life (18). There is an increased frequency of tracheitis and bronchitis in infants whose parents smoked, most of the risk is derived from maternal smoking (19).

A study of random community-based populations in Michigan and Massachusetts showed that children of smokers were more likely to have asthma, particularly severe asthma, than children of nonsmoking parents (20). In a study of children aged 7 to 17 years with a history of asthma, the children of mothers who smoked had 47% more symptoms, a lower FEV1 (13% lower), and a fourfold greater responsiveness to a histamine challenge test than asthmatic children of nonsmoking mothers (21). Additionally, a study of asthmatic children showed a significant increase of 63% in asthma-related visits to an ER by children exposed to parental tobacco smoke (22). The published data to date suggest that parental smoking, particularly maternal smoking, increases the frequency and severity of asthmatic attacks (23).

### Interventions in the Inner City

Too few interventions have been tried in patients in urban centers. The long-term effectiveness of education in modifying behavior is unknown; but it is likely that education alone of patients and their families, especially about the importance of preventive care, will significantly reduce mortality and morbidity. Measures to reduce exposure to environmental allergens and irritants and to eliminate some of the barriers to access to health care are likely to make a major positive impact.

The roles of education and avoidance of allergens and irritants cannot be overemphasized. Children with asthma have hyperresponsive airways, and the need to understand the chronicity of this disease will avoid the episodic approach to both nonpharmacologic and pharmacologic management. Education involves helping families understand asthma and learn and practice skills necessary to manage the illness. Providing information (written or verbal) is necessary, but is not sufficient to accomplish these objectives. Education must begin at the time of diagnosis and continue with each office visit and telephone call. Each office visit, however brief, should be viewed as an opportunity for patient and family education. The educational needs of patients and families may change with time and should be assessed at regular intervals. Encouraging active participation in a part-

nership with the clinician will improve environmental control measures and patient adherence to a management plan.

Avoidance of allergens and irritants that produce airway narrowing is important since these factors are known to provoke acute symptoms and increase airway hyperresponsiveness. This, in turn, increases vulnerability to further irritant and allergen exposure. If allergy plays a role in a patient's asthma symptoms, environmental control measures to avoid specific allergens are of paramount importance, and immunotherapy may be indicated in selected patients.

Central to the health effects of indoor allergens and the importance of education is the role of allergen avoidance as a primary method of promoting good health and controlling diseases initiated or exacerbated by exposure to these allergens. For example, mite allergen avoidance is considered an important method of treatment for dust mite allergy (24) and is also associated with improvement of asthma when rigorous methods of avoidance are employed (25). In the Canadian study by Murray and Ferguson there were substantial reduction in bronchial hyperreactivity and medication requirements in ten children who used mite avoidance measures. The regimen was stringent and included removing carpets, sealing heating ducts, and removing from the home animals to which the children had positive allergy skin tests (26).

Studies suggest that allergen avoidance reduces morbidity of asthma in sensitized individuals and that there is correlation of high prevalence of immediate hypersensitivity to common indoor allergens and existence of threshold levels of exposure in individuals at risk. Recent progress in the immunochemical detection of common indoor allergens such as cat, dust mite, and cockroaches makes it possible to measure exposure to these allergens and to define the threshold levels of exposure that are needed to effect sensitization and increased symptoms (27). For example, it has been suggested that exposure to greater than 2 µg group I dust mite allergen (or 100 mites) per gram of dust increases the risk of children developing sensitization and asthma. In addition to the risk of sensitization to allergens derived from cats, cockroaches, and grass pollen, endotoxins are strongly suspected as potent pro-inflammatory substances in individuals exposed to high levels of these allergens (28). Finally, in a prospective study in a cohort of British children at risk of allergic disease because of

family history, there was a trend toward sensitization to dust mites by age 11 when exposure to antigen at age 1 had been to more than 10 µg Der p 1/g of dust (29). The age at which wheezing first occurred was positively related to the level of exposure at age 1 for all children, but especially for those who had a family history of atopy. In this study it is suggested that in addition to genetic factors, exposure to certain allergens in early childhood may be an important determinant for subsequent development of respiratory diseases such as asthma. Thus, avoidance of indoor allergens may be important in managing asthma and will likely be significant in reducing morbidity in susceptible individuals.

Future studies are necessary to answer the questions of how stringent and for how long avoidance measures should be attempted, since not all studies have been uniformly positive in demonstrating the beneficial effect of allergen avoidance on allergic diseases (30). Among 26 children with mild to moderate asthma who participated in a controlled trial of dust mite avoidance for up to 12 weeks, there was no significant difference among the study and

control groups in bronchial reactivity to histamine, symptom scores, peak expiratory flow rates, or medication requirements. However, the measures of avoidance were less stringent than those of the studies cited above. On the other hand, it is argued that it may be difficult to convince parents of children with mild to moderate asthma (as opposed to severe asthma) that more stringent methods are warranted. While this study failed to show clinical benefit from mite avoidance, there was a fall in total serum IgE, the significance of which is unknown at present. The long-term effects of avoidance and the appropriate patient population on whom such measures may be effective are presently unknown.

In some cases objective monitoring of lung function may be an important means of monitoring exposure to allergens and irritants. Peak Expiratory Flow Rates (PEFR) have been reported to be useful in daily monitoring to detect early stages of airway obstruction and as a guide to initiate therapy; in determining when emergency care is needed; and in obtaining daily measures to investigate the potential role of

allergens at home or in the workplace. Further studies are needed to determine which group of patients (e.g., by severity of asthma) or under what circumstances monitoring of PEFR will be effective.

## Summary

Studies to date suggest that indoor allergens and irritants play a significant role in level of asthma morbidity experience by children living in urban centers. Although relatively few controlled studies have been done to assess the significance of and to intervene in reducing exposure to environmental allergens (especially indoor allergens) and irritants in this population, the following suggestions can be made:

- provide education to patients and health care providers about environmental risk factors (allergens and irritants), especially in urban centers;
- stress prevention of asthma symptoms and promote days of wellness and normal function;
- initiate and maintain surveillance of individuals and groups who are at risk of exposure to high levels of allergens and irritants.

## REFERENCES

1. Malveaux FJ. Deaths from asthma by race, sex, and age. 1979–83. *J Allergy Clin Immunol* 79:183 (1987).
2. Wissow L, Gittelsohn A, Szklo M, Starfield B, Mussman M. Poverty, race, and hospitalization for childhood asthma. *Am J Public Health* 78:777–782 (1988).
3. Halfon N, Newacheck PW. Childhood asthma and poverty: differential impacts and utilization of health services. *Pediatrics* 91:59–61 (1993).
4. Marder D, Targonsky P, Orris O, Persky V, Addington W. Effect of racial and socioeconomic factors on asthma mortality in Chicago. *Chest* 101:427S–430S (1992).
5. Carr W, Zeitel L, Weiss K. Asthma hospitalization and mortality in New York City. *Am J Public Health* 82:59–65 (1992).
6. Data Sourcebook: Five Million Children. New York: Columbia University National Center for Children in Poverty 1990;9.
7. Neighbors HW. Ambulatory medical care among adult Black Americans: the hospital emergency room. *J Natl Med Assn* 78:275–282 (1986).
8. Hubbell F, Waitzkin H, Mishra S, Dombrink J. Evaluating health care needs of the poor: a community-oriented approach. *Am J Med* 87:127–131 (1989).
9. Rea HH, Scragg R, Jackson R, Beaglehole R, Fenwick J, Sutherland, DC. A case-controlled study of deaths from asthma. *Thorax* 41:833–839 (1986).
10. Bernton H, Brown H. Insect allergy: preliminary studies of the cockroach. *J Allergy* 35:6 (1964).
11. Fletcher-Vincent S, Reece ER, Malveaux FJ. Reactivity to cockroach and other allergens in an inner city population with rhinitis and asthma. *J Allergy Clin Immunol* 93:174 (1994).
12. Kang B. Study on cockroach antigen as probable causative agent in bronchial asthma. *J Allergy Clin Immunol* 58:357–365 (1976).
13. Kang B, Vellody D, Homburger H, Yunginger JW. Cockroach cause of allergic asthma: its specificity and immunologic profile. *J Allergy Clin Immunol* 63:80–86 (1979).
14. Cockcroft D. *Lancet* 1:253 (1983).
15. Friedman GD, Pettiti DB, Bawol RD. Prevalence and correlates of passive smoking. *Am J Public Health* 73:401–405 (1983).
16. Weiss ST. Passive smoking and lung cancer: what is the risk? *Am Rev Respir Dis* 133:1–3 (1986).
17. Harlap S, Davies AM. Infant admissions to hospital and maternal smoking. *Lancet* 1:529–532 (1974).
18. Colley JR, Holland WW, Corkhill RT. Influence of passive smoking and parental phlegm on pneumonia and bronchitis in early childhood. *Lancet* 2:1031–1034 (1974).
19. Pedreira FA, Guandolo VL, Ferali EJ, Mella GW, Weiss LP. Involuntary smoking and incidence of respiratory illness during the first year of life. *Pediatrics* 75:594–597 (1985).
20. Gortmaker SL, Walker DK, Jacobs FH, Ruck-Ross H. Parental smoking and the risk of childhood asthma. *Am J Public Health* 72(6):574–579 (1982).
21. Murray AB, Morrison BJ. The effect of cigarette smoke from the mother on bronchial responsiveness and severity of symptoms in children with asthma. *J Allergy Clin Immunol* 77(4):575–581 (1986).
22. Evans D, Levison MJ, Feldman H, Clark NM, Wasilewski Y, Levin B, Mellins RB. The impact of passive smoking on emergency room visits of urban children with asthma. *Am Rev Respir Dis* 135:567–572 (1987).
23. Fielding JE, Phenow RJ. Health effects of involuntary smoking. *N Engl J Med* 319:1452–1460 (1988).
24. Buckley JM, Pearlman DS. Controlling the environment for allergic diseases. In: *Allergic Diseases from Infancy to Adulthood*. 2nd ed (Bierman CW, Pearlman DS, eds). Philadelphia:W.B. Saunders Company, 1989;239–252.
25. Platts-Mills T, Tovey EB, Mitchell EB, Moszoro H, Nock P, Wilkins SR. Reduction of bronchial hyperreactivity during prolonged allergen avoidance. *Lancet* 2:675–678 (1982).
26. Murray AB, Ferguson AC. Dust-free bedrooms in the treat-

- ment of asthmatic children with house dust or house dust mite allergy: a controlled trial. *Pediatrics* 71:418–422 (1983).
27. Platts-Mills T, Ward GW, Sporik R, Gelber L, Chapman MD, Heymann P. Epidemiology of the relationship between exposure to indoor allergens and asthma. *Int Arch Allergy Appl Immunol* 94:339–345 (1991).
  28. Michel O, Ginanni R, Duchateau J, Vertongen F, Le Bon B, Sergysels R. Domestic endotoxin exposure and clinical severity of asthma. *Clin Exp Allergy* 21:441–448 (1991).
  29. Sporik R, Holgate ST, Platts-Mills T, Cogswell J. Exposure to house dust mite allergen (Der p I) and the development of asthma in childhood: a prospective study. *N Engl J Med* 323:502–507 (1990).
  30. Gillies DRN, Littlewood JM, Sarsfield JK. Controlled trial of house dust mite avoidance in children with mild to moderate asthma. *Clin Allergy* 17:105–111 (1987).